CLINICAL VIGNETTE

Caution! Treating Hansen's Disease In The Face Of Hepatitis B

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Introduction

Hepatitis B virus (HBV) reactivation is an increasingly recognized complication of immunosuppressive biological agents[1]. However, reactivation due to systemic glucocorticoids is rarely reported in the dermatologic literature. We report a patient who developed HBV reactivation, and subsequently liver failure, following glucocorticoid treatment for Hansen's disease.

Case Report

A 59-year-old Filipino immigrant with chronic HBV presented for orthotopic liver transplantation (OLT) evaluation. He developed liver failure, following treatment for Hansen's disease. This was diagnosed several months earlier, when the patient presented to an outside dermatology clinic with a one-year history of hyperpigmented lesions on his trunk and extremities. Skin biopsies were consistent with leprosy. Fite, Ziehl-Neelsen, and Flourostain stains were positive for the mycobacteria. He was initially started on clarithromycin and prednisone and Rifampin and ethambutol were subsequently added. Routine laboratory studies two months later demonstrated marked transaminitis and hyperbilirubinemia. Three months after treatment initiation, he developed weakness and jaundice and was found to be in liver failure.

The patient was subsequently transferred to our institution for OLT evaluation. Skin exam on admission demonstrated numerous, well-marginated, annular, hyper-pigmented, and symmetrically distributed patches and several flesh-colored papules consistent with the tuberculoid and histioid leprosy diagnosis. There was no alopecia nor diminished sensation within the lesions (Figure 1). Admission laboratory studies revealed an aspartate aminotransferase of 513 U/L, alanine aminotransferase of 1299 U/L, total bilirubin of 34.8 mg/dL, and HBV viral load of 12900 IU/mL. Liver biopsy demonstrated patchy necrosis and evidence of active and chronic HBV.

It was felt that the immunosuppression from the prednisone used to treat Hansen's Disease, caused HBV reactivation and con-sequent hepatic decompensation. The prednisone was rapidly tapered and antiviral medications started. Due to the patient's active Hansen's Disease, he was not considered a candidate for OLT and he died ten days later.

Reactivation of latent HBV secondary to systemic glucocorticoid therapy is rarely reported. Yang et al[2] reported four cases of HBV reactivation out of 98 patients receiving long-term prednisolone therapy for pemphigus vulgaris and dermatomyositis. Cheng et al[3] demonstrated in a multi-center randomized clinical trial that patients receiving chemotherapy with concomitant glucocorticoids had significantly increased rates and severity of HBV reactivation compared to patients undergoing chemotherapy without glucocorticoids. Notably, these studies were conducted in Taiwan where the prevalence of HBV is 10%[4]. In the U.S., the prevalence of HBV is only 0.5%. However, immigrants from Asia and developing countries are considered high risk for HBV infection and a large proportion of Hansen's disease patients in the U.S. are from these countries[4,5].

Given the increasing reports of HBV reactivation in patients receiving systemic glucocorticoids, it may be useful to screen for HBV infection and to consult a gastroenterologist for patients with history of prior infection or underlying chronic infection.

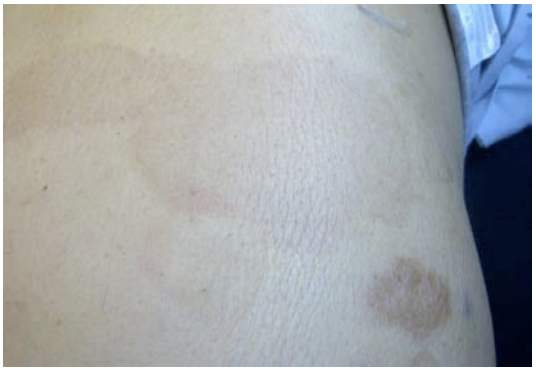

FIGURE 1; note the well-defined erythematous plaque on the chest

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